

**U.S. Department of Commerce  
Patent and Trademark Office**

<b>Application Number</b>	10/666,513
<b>Filing Date</b>	September 19, 2003
<b>First Named Inventor</b>	Ann Marie Schmidt
<b>Art Unit</b>	1646
<b>Examiner Name</b>	J. Andres
<b>Attorney Docket No.</b>	55873-C/JPW/AJM/JCS

**(Use several sheets if necessary)**

[illegible][illegible]

Gregory Lund

6/24/05

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<b>Form PTO-1449</b> <b>U.S. Department of Commerce</b> <b>Patent and Trademark Office</b>  <b>INFORMATION DISCLOSURE CITATION</b> (Use several sheets if necessary)	<b>Application Number</b>	10/666,513
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**NON PATENT LITERATURE DOCUMENTS**

Examiner Initials <sup>2</sup>	Cite No. <sup>1</sup>	Include name of the author (in CAPITAL LETTERS), title of the article (when appropriate), title of the item (book, magazine, journal, serial, symposium, catalog, etc.) date, page(s), volume-issue number(s), publisher, city and/or country where published.	T <sup>2</sup>
HA		March 24, 2005 Communication from the European Patent Office transmitting a Supplementary Partial European Search Report Under Rule 45 EPC in connection with European Patent Application No. 99953081.9, filed October 6, 1999	
		Miyata, T., et al., "The receptor for advanced glycation end products (RAGE) is a central mediator of the interaction of AGE- $\beta_2$ microglobulin with human mononuclear phagocytes via an oxidant-sensitive pathway: Implications for the pathogenesis of dialysis-related amyloidosis," Journal of Clinical Investigation 98: 1088-1094 (1996)	
		Hori, O., et al., "The receptor for advanced glycation end-products has a central role in mediating the effects of advanced glycation end-products of the development of vascular disease in diabetes mellitus," Nephrology Dialysis Transplantation 11: 13-16 (1996)	
		Li, J., et al., "Characterization and functional analysis of the promoter of RAGE, the receptor for advanced glycation end products," Journal of Biological Chemistry 272: 16498-16506 (1997)	
		Hofmann, M., et al., "EN-RAGE (extracellular novel-RAGE binding protein) activates endothelial cells and macrophages to mediate inflammatory responses," Circulation 98: 1316 (1998)	

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